

The listing of claims will replace all prior versions, and listings, of claims in the application.

Claims 1-31. (canceled)

Claim 32. (currently amended) A method for predicting the pharmacological effect a drug candidate compound would have in a cell, tissue, or organ that expresses a protein, comprising:

- a) modulating, by somatic gene transfer, expression of the protein in host cells;
- b) comparing the phenotype of the host cells in which expression of the protein has been modulated to the phenotype of control host cells in which expression of the protein has not been modulated; and
- c) analyzing the result of expression of the protein by correlating the result of expression of the modified protein to the result of expression of a corresponding native protein, wherein a result of expression of the protein whose expression modulated in step (a) may mimics one or more of the effects of the drug candidate compound, and wherein the phenotype is propagation of an electrical charge, pattern of electrical signaling, contraction, growth, blebbing or budding, pycnotic transformation, kinesis, cell death, differentiation, replication, transcription, translation, protein processing, adhesion, oncogenetic transformation, enzymatic catalysis, or functional modification.

Claim 33. (previously presented) The method of claim 32, wherein the protein is a drug target protein.

Claim 34. (previously presented) The method of claim 32, wherein the difference in phenotype between the host cells in which expression of the protein has been modulated and the phenotype of control host cells in which expression of the protein has not been modulated comprises an alteration in a function of the cells.

Claim 35. (previously presented) The method of claim 32, wherein the difference in phenotype between the host cells in which expression of the protein has been modulated and the phenotype of control host cells in which expression of the protein has not been modulated comprises suppression of a function of the cells.

Claim 36. (previously presented) The method of claim 32, wherein the difference in phenotype between the host cells in which expression of the protein has been modulated and the phenotype of control host cells in which expression of the protein has not been modulated comprises induction of a function of the cells.

Claim 37. (cancelled).

Claim 38. (previously presented) The method of claim 32, wherein expression of the protein is increased following the somatic gene transfer.

Claim 39. (previously presented) The method of claim 38, wherein the increase in expression is achieved by operably linking a gene encoding the protein to an inducible or viral promoter.

Claim 40. (previously presented) The method of claim 32, wherein expression of the protein is inhibited following the somatic gene transfer.

Claim 41. (previously presented) The method of claim 40, wherein expression of the protein is inhibited by transfer of a gene truncated relative to a corresponding native gene.

Claim 42. (previously presented) The method of claim 41, wherein the truncation is a contiguous or non-contiguous deletion of the transferred gene.

Claim 43. (previously presented) The method of claim 42, wherein expression of the protein is inhibited by transfer of a gene encoding one or more amino acid substitutions relative to a corresponding native protein.

Claim 44. (previously presented) The method of claim 32, wherein the protein is capable of specifically forming a binding complex with at least one other protein molecule.

Claim 45. (previously presented) The method of claim 44, wherein expression of the protein is sufficient to produce a dominant negative mutation that reduces or blocks function of the binding complex.

Claim 46. (previously presented) The method of claim 32, further comprising screening the potential drug target protein using natural products testing, synthetic chemical testing, combinatory chemistry, targeted diversity, rational drug design, or selective gene suppression techniques.

Claim 47. (previously presented) The method of claim 32, wherein the phenotype is propagation of an electrical charge.

Claim 48. (previously presented) The method of claim 32, wherein the phenotype is cell growth.

Claim 49. (previously presented) The method of claim 32, wherein the phenotype is blebbing or budding.

Claim 50. (previously presented) The method of claim 32, wherein the phenotype is pycnotic transformation.

Claim 51. (previously presented) The method of claim 32, wherein the phenotype is kinesis.

Claim 52. (previously presented) The method of claim 32, wherein the phenotype is cell death.

Claim 53. (previously presented) The method of claim 32, wherein the phenotype is cell differentiation.

Claim 54. (previously presented) The method of claim 32, wherein the phenotype is cell replication.

Claim 55. (previously presented) The method of claim 32, wherein the phenotype is transcription or translation.

Claim 56. (previously presented) The method of claim 32, wherein the phenotype is protein processing.

Claim 57. (previously presented) The method of claim 32, wherein the phenotype is protein adhesion.

Claim 58. (previously presented) The method of claim 32, wherein the phenotype is oncogenetic transformation.

Claim 59. (previously presented) The method of claim 32, wherein the phenotype is enzymatic catalysis.

Claim 60. (previously presented) The method of claim 32, wherein the phenotype is protein modification.

Claim 61. (previously presented) The method of claim 32, wherein the phenotype is pattern of electrical signaling.

Claim 62. (previously presented) The method of claim 32, wherein the phenotype is contraction.

Claim 63. (previously presented) The method of claim 32, wherein the phenotype is cell growth.

Claim 64. (previously presented) The method of claim 32, wherein the phenotype is functional modification.